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Non-contrast–enhancing subdural empyema: illustrative case

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BACKGROUND Subdural empyema (SDE) is a life-threatening intracranial infection that, without timely surgical intervention and appropriate antibiotic treatment, is inevitably fatal. SDE is classically recognized on brain imaging as a subdural collection surrounded by a contrast-enhancing ring.

OBSERVATIONS The authors describe the case of a 41-year-old male with clinical features consistent with SDE but without any contrast enhancement on multiple computed tomography scans obtained more than 48 hours apart. Given the high clinical suspicion for SDE, a craniotomy was performed that demonstrated frank pus that eventually grew *Streptococcus pyogenes*.

LESSONS This case demonstrates that SDE may present without ring enhancement on contrast-enhanced imaging. In critically ill patients with a high clinical suspicion for SDE despite lack of contrast enhancement, we demonstrate that exploratory burr holes or craniotomy can provide diagnostic confirmation and source control.

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KEYWORDS subdural empyema; brain abscess; cerebritis; infection

Subdural empyema (SDE) is a life-threatening intracranial infection between the dura and arachnoid mater.[1] Without timely surgical intervention and appropriate antibiotics, SDE is inevitably fatal. However, with prompt intervention, mortality ranges from 6% to 35%.[1,2] The imaging findings of SDE classically describe a subdural collection surrounded by a membrane that avidly enhances.[1,3] It is more common in males and common etiologies include sinusitis, otitis media, hematological spread from distant sites, cranial surgery, trauma, and meningitis in children.[1,2] Herein, we present the diagnostically challenging case of a 41-year-old male presenting with a non-contrast–enhancing SDE.

Illustrative Case

A 41-year-old male with a history of polysubstance use presented to hospital with confusion, agitation, and headache after having inhaled heroin. He denied any constitutional symptoms, recent travel, or sick contacts, but reported recurrent falls in the past 6 weeks, including a fall through a glass table that cut his hand and required sutures.

On admission, he was febrile at 38.2°C and his Glasgow Coma Scale (GCS) score was 11 (E3/V3/M5). He had a superficial skin infection on his hand. Neurologically, he had nuchal rigidity and a left gaze preference. Following painful stimulus, he demonstrated left arm flexor posturing and right arm extensor posturing. Laboratory investigations revealed significant leukocytosis (white blood cell [WBC] 24.1 $\times$ 10^9/L), an elevated serum lactate (2.3 mmol/L), and an elevated C-reactive protein (258 mg/L). Contrast-enhanced computed tomography (CT) of the head revealed a nonenhancing hypodense left parietal subdural collection with adjacent effacement of the sulci but without any brain edema, herniation, or midline shift (Fig. 1). This was interpreted as most likely a chronic subdural hematoma given his recent falls. Dedicated vascular imaging (CT angiography) did not demonstrate any vessel occlusions or stenosis. A lumbar puncture was deferred since

ABBREVIATIONS CT = computed tomography; GCS = Glasgow Coma Scale; MRI = magnetic resonance imaging; SDE = subdural empyema; WBC = white blood cell count.

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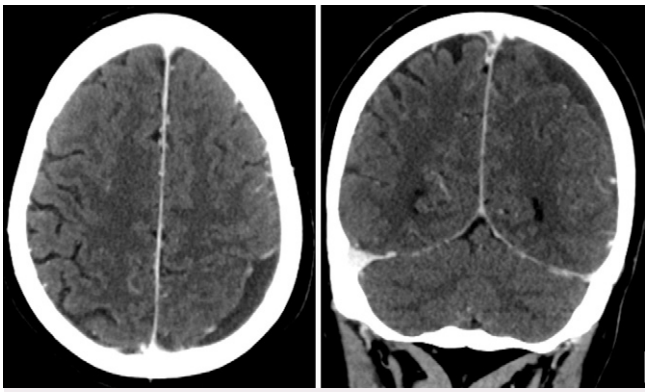

FIG. 1. Axial (**left**) and coronal (**right**) contrast-enhanced CT of the head demonstrating a nonenhancing left parietal subdural collection.

international normalized ratio and partial thromboplastin time were elevated.

The patient was admitted to the medicine ward and ceftriaxone, vancomycin, and acyclovir were begun for empirical treatment of bacterial or viral meningoencephalitis, as well as phenytoin for suspected seizures. Chest radiography, blood and urine cultures, transthoracic echocardiography, and urine analysis were unremarkable. He was awaiting cranial magnetic resonance imaging (MRI). On the second day of admission his fevers and leukocytosis persisted, while his GCS score deteriorated to 9 (E2/V3/M4) with left arm withdrawal and right arm extensor posturing with painful stimuli. This necessitated transfer to the intensive care unit. Repeat head CT with and without contrast demonstrated a stable appearance of the subdural collection without any enhancement or brain edema.

Given the patient's ongoing sepsis and acute clinical deterioration, there was a high clinical suspicion that his parietal lesion was in fact an SDE. Therefore, the patient was taken urgently to the operating room for an exploratory burr hole procedure. The burr hole revealed frank pus (Fig. 2). Therefore, a craniotomy was performed and his subdural empyema was evacuated. Intraoperative cultures grew *Streptococcus pyogenes*. Postoperatively he remained on intravenous antibiotics (ceftriaxone and metronidazole) for 6 weeks. Five weeks after his initial presentation, he was transferred to a rehabilitation hospital with mild right upper extremity weakness and language deficits, otherwise he was neurologically well.

Discussion

Observations

Rapid recognition and prompt evacuation of SDE is imperative to improve the chance of survival with good neurological recovery. The diagnostic test of choice for intracranial infections, including SDE, is a contrast-enhanced MRI with gadolinium.[1] However, MRI may not be readily available in all centers, notably in low-resource settings, and in unstable patients contrast-enhanced CT may be more appropriate.[1] Regardless of imaging modality, SDE is classically described as a subdural collection surrounded by a membrane that avidly enhances, particularly along the medial surface.[1,3] Pathophysiologically, this is thought to represent an inflammatory process through which the body encapsulates the collection of pus, allowing for permeability to contrast agent.[4] Early cerebritis, occurring 1 to 3 days postinfection, has been described to be nonenhancing on CT and MRI.[5,6] However, with both

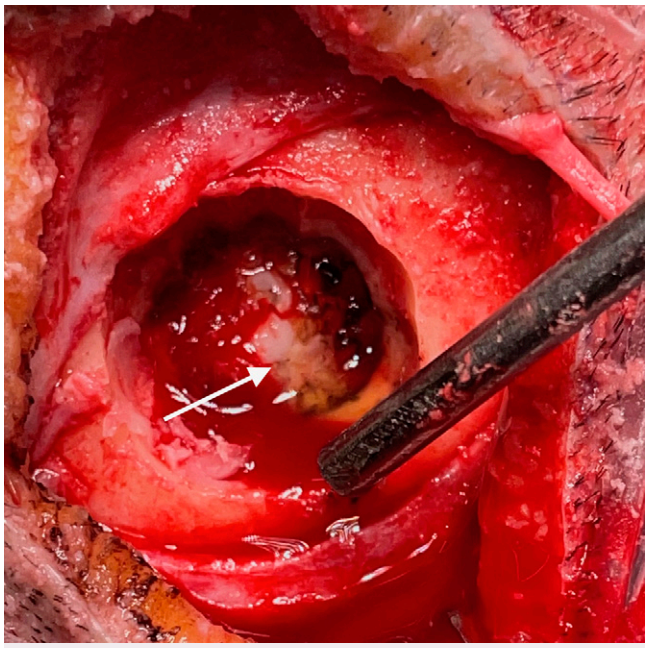

FIG. 2. Intraoperative image obtained after a burr hole procedure, demonstrating pus (*arrow*).

early cerebritis and untreated SDE, edema of the adjacent cortex typically occurs, which was not seen in our case.[5,6] In cases with diagnostic uncertainty MRI may be helpful, specifically, diffusion-weighted imaging sequences demonstrate restricted diffusion in SDE due to the viscosity of the purulent nature of the empyema.[7]

Initial CT descriptions of SDE noted that lack of enhancement does not exclude an acute empyema, and they described three cases of small acute empyemas lacking marginal contrast enhancement.[3,4] These studies were from the 1980s when CT scanners were much less powerful than they are today, and the authors did not publish their images. Although our patient was neither immunocompromised or immunosuppressed and had two serial CT scans demonstrating lack of ring enhancement, the presumption would be that a lack of the usual inflammation-related permeability of the blood-brain barrier was the cause of the lack of enhancement.

The fulminant clinical course of SDE consists of fever, lethargy, meningism, and headache followed by the rapid development of seizures, hemiparesis, and coma.[1,3] Our patient followed this clinical course even while on broad-spectrum antibiotics and antivirals, which raised the clinical suspicion for SDE even without the classic appearance of avid ring enhancement on CT. Given the importance of early and aggressive surgical evacuation for reducing the degree of cortical damage and subsequent neurological deficits with SDE, clinicians should not delay treatment in order to obtain further diagnostic tests.[1,3] If the patient, like ours, is critically ill and there is a high clinical suspicion for SDE, we advocate for exploratory burr hole procedures or craniotomy. This allows for diagnostic confirmation and source control.

SDEs are often polymicrobial; thus, initial antibiotic treatment should include broad-spectrum coverage directed against common causative organisms, including anaerobic gram-positive cocci, *Streptococcus* and *Staphylococcus* species, and anaerobic gram-negative bacilli.[1,8] Recommended empirical antibiotic regimes include

a third-generation cephalosporin, vancomycin, and metronidazole, since these offer broad coverage with good cerebrospinal fluid and abscess penetration.[1,8] After consultation with infectious diseases, antibiotics may be tailored if an organism is identified. Cultures from surgical specimens may be negative in up to 50% of patients with SDE.[1,8] Intravenous antibiotics should be continued for a minimum of 2 weeks, followed by parenteral or oral antibiotics for a total 6-week treatment duration.[8]

Lessons

This case demonstrates that SDE may present without ring enhancement on contrast-enhanced imaging. Our patient's subdural collection was nonenhancing on two separate CT scans nearly 48 hours apart, which made the case a diagnostic challenge. The key clinical feature was his rapidly deteriorating clinical course while on broad-spectrum antimicrobials, which in the context of a subdural collection, was highly concerning for a potential SDE. Given the importance of early and aggressive surgical evacuation for reducing the degree of cortical damage and subsequent neurological deficits with SDE, exploratory burr hole procedures or craniotomy may be performed in critically ill patients with a high clinical suspicion for SDE.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Rebchuk, Griesdale. Acquisition of data: Rebchuk. Analysis and interpretation of data: Rebchuk, Griesdale. Drafting the article: Rebchuk, Griesdale. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Rebchuk. Study supervision: Griesdale, Honey.

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